Training improves the speed of aimed movements in Parkinson’s disease

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Summary
In this study, the extent to which bradykinesia in patients with idiopathic Parkinson’s disease can be influenced by practice and by specific training strategies was investigated. Fifteen patients with Parkinson’s disease tested after withdrawal of anti-Parkinson medication, and 15 matched control subjects, practised a ballistic aiming task. Performance was tested before, during and after training and again 1 h later. The Parkinson’s disease patients and control subjects were randomly assigned to one of two training schedules, practising with or without rhythmic auditory cues. At baseline, the Parkinson’s disease patients showed longer movement times, with a marked decrease in maximum acceleration and deceleration in the initial open-loop phase compared with those of the control subjects. With training, they were able to make significant improvement in the speed of aimed movements, particularly in the early movement phase, without any deterioration in accuracy. These effects transferred to an untrained limb and were at least partially maintained after a 1-h delay.

While patients remained impaired relative to control subjects at all phases of training and follow-up, the patients’ performance at the end of training did not differ significantly from the control subjects’ baseline function. Contrary to expectation, rhythmic auditory cues did not enhance improvement in the speed of aimed movements in either patients or control subjects. If anything, less improvement was shown in the cued groups, although there were suggestions that the aiming skill was retained better over the delay period. The results demonstrate preserved abilities to improve speed of single ballistic aiming movements in Parkinson’s disease patients and the possibility of reducing bradykinesia by training.

Keywords: motor learning; skill; arm; movement; striatum

Abbreviations: IT = immediate testing (immediately after training); DT = delayed testing (1 h after training)

Introduction
Bradykinesia is the cardinal sign of Parkinson’s disease thought to reflect most closely the underlying striatal dopamine depletion. Mechanisms underlying motor slowness, however, are only partly understood, and little is known about how bradykinesia may be influenced by training, in spite of its potential clinical therapeutic value. It had been noted 70 years ago that even the simplest voluntary movements in Parkinson’s disease are delayed both in initiation and execution (Wilson, 1925). While deficits are found in movement preparation (Jahanshahi et al., 1992), the predominant impairment appears to be movement execution (Day et al., 1984; Rafal et al., 1989; Stelmach et al., 1989). Slower movements in Parkinson’s disease are associated with multiple bursts of agonist electromyographic activity instead of a large initial burst (e.g. Hallett and Khoshbin, 1980; Teasdale et al., 1990) suggesting a problem in the control of muscle activation and its regulation over time.

Although bradykinesia is a fundamental aspect of Parkinson’s disease, and it contributes significantly to the overall handicap resulting from the disease, there have been relatively few attempts to assess whether it can be improved by practice and specific training techniques. A few clinical reports, providing limited information, have suggested that physical therapy could be beneficial (e.g. Doshay, 1962; Eni, 1988; Schenkman et al., 1989; Comella et al., 1994). However, such reports are provocative, as there is evidence that motor learning may be impaired in Parkinson’s disease. Functional brain imaging studies suggest that the putamen
and/or its associated cortical projection areas, including the supplementary motor area, may form parts of a functional system subserving motor learning (Seitz et al., 1990; Friston et al., 1992; Grafton et al., 1992). Patients with Parkinson’s disease have been shown to benefit from practice in some motor tasks (e.g. Day et al., 1984; Robertson and Flowers, 1990; Worringham and Stelmach, 1990), although often more slowly than in normal subjects. However, others have demonstrated impaired learning in patients with Parkinson’s disease on tasks such as manual pursuit and sequence learning (Frith et al., 1986; Heindel et al., 1989; Harrington et al., 1990; Jackson et al., 1995; Soliveri et al., 1997). To date, however, no study has investigated the sensitivity of movement speed to practice and training.

In the present study, a visually guided aiming movement was chosen as the paradigm. Such tasks have been used previously to demonstrate bradykinesia in patients with Parkinson’s disease. Sheridan and Flowers (1990) illustrated that patients with Parkinson’s disease have difficulty in making such movements both quickly and accurately, resorting instead to a slower visually guided movement that ensured end-point accuracy. The implication from that paper was that patients could be relatively fast, or accurate, but not both. One advantage of such tasks is that kinematic analysis enables the assessment of spatial, temporal and force-related aspects of movements and their possible changes during an experimental session. This provides a much more detailed assessment of the behaviour change than the more global accuracy-based measures typically employed in motor learning studies.

The primary aims of the study were to determine (i) the effects of practice on these correlates of bradykinesia, and (ii) the degree to which any improvement transferred both across time and to an untrained limb. In addition, we sought to determine whether training was sensitive to the provision of external rhythmic cues to movement speed. Such cues help patients to sustain rhythmic motor activity (Freeman et al., 1993) and have been used practically in physiotherapy to improve gait pattern with Parkinson’s disease patients (Eni, 1988). It has been suggested that such cues utilize additional cerebellar and cortical areas to control movement and thus minimize the impact of dysfunction in the system involved in the control of self-initiated movements (Jahanshahi et al., 1995). Because this latter system may also be involved in motor learning, as discussed above, auditory cues may also enhance learning by recruiting additional cerebral regions in the process.

**Methods**

**Subjects**

Sixteen patients with clinically defined Parkinson’s disease were recruited from out-patient clinics at the National Hospital for Neurology and Neurosurgery, London, UK. All were right handed as defined by the Edinburgh Handedness Inventory (Oldfield, 1971), and none had marked tremor, dyskinesia or susceptibility to sudden ‘off’ (medication) periods. Subjects were excluded if they had any other disabling general medical condition or concomitant nervous diseases including dementia. Subjects were randomly assigned to one of the two training conditions, with and without auditory rhythmic cues (see below). One patient was subsequently excluded due to markedly poor performance, uncharacteristic of the other patients in the sample. Each patient was paired with a sex-matched control subject of approximately the same age. All control subjects were right handed. None reported a history of neurological or musculoskeletal problems, and none had neurological signs at the time of testing. No control subject was taking drugs that affect the CNS.

Clinical assessments for patients and control subjects included a standardized neurological examination and the Mini-Mental Status Examination (DePaulo, 1980), and Beck Depression Inventory (Beck et al., 1961). The patients’ disease-related motor impairment was assessed with the Columbia Rating Scale and the modified Hoehn and Yahr staging according to the United Parkinson’s Disease Rating Scale V (Lang and Fahn, 1989). During the experimental session, bradykinesia was monitored by asking subjects to perform a repetitive thumb flexion task at maximal rate for 30 s and using a tally-counter. This task was performed with the more affected arm before training, immediately after training and again after the delay period.

The characteristics of the subjects are given in Table 1, where they are divided into the two experimental subgroups. While patients in subgroup I had a longer disease duration ($P < 0.05$; Mann–Whitney $U$ test), neither symptom severity nor Hoehn and Yahr rating differed significantly. Overall, the four subgroups (cued or uncued condition, patient or control subject) were matched for both age and Mini-Mental Status Examination score. There was a tendency for the patients to score higher on the Beck Depression Inventory [$F(1,26) = 3.26, P = 0.08$], although the two subgroups did not differ [$F(1,26) < 1$].

Each patient’s more bradykinetic arm was identified and used during the training trials. Subsequent transfer effects were measured on the less affected arm (see below). Control subjects used the same limb as their pair-matched patient. All patients were in a clinically defined ‘off’ state at the time of the experiment, and had taken their last antiparkinsonian medication $\geq 12$ h before the start of the study session.

The study was approved by the ethical committee of the National Hospital for Neurology and Neurosurgery. Informed consent was obtained from each subject.

**Procedure**

A digitizing tablet (active area $\sim 457\times305$ mm) (Model RDT1218, Scriptel Corp., Columbus, Ohio, USA) with a sampling rate of 100 Hz and spatial resolution of 0.025 mm was used for the aiming task. Experimental control and on-line data collection were carried out using an IBM-
Table 1 Group characteristics

<table>
<thead>
<tr>
<th></th>
<th>Parkinson’s disease patients</th>
<th></th>
<th>Control subjects</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Subgroup I (n = 7)</td>
<td>Subgroup II (n = 8)</td>
<td>Subgroup I (n = 7)</td>
<td>Subgroup II (n = 8)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>65.9 ± 8.3</td>
<td>62.0 ± 14.6</td>
<td>62.1 ± 13.3</td>
<td>60.8 ± 15.2</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>3/4</td>
<td>5/3</td>
<td>3/4</td>
<td>5/3</td>
</tr>
<tr>
<td>Trained arm (R/L)</td>
<td>3/4</td>
<td>5/3</td>
<td>3/4</td>
<td>5/3</td>
</tr>
<tr>
<td>MMSE</td>
<td>27.7 ± 1.6</td>
<td>28.8 ± 1.0</td>
<td>28.0 ± 1.4</td>
<td>27.6 ± 1.6</td>
</tr>
<tr>
<td>Duration of illness (years)</td>
<td>7.6 ± 2.4</td>
<td>4.3 ± 1.8</td>
<td>6.5 ± 7.6</td>
<td>4.5 ± 2.5</td>
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<tr>
<td>Hoehn and Yahr stage</td>
<td>2.5 ± 0.5</td>
<td>2.0 ± 0.75</td>
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<tr>
<td>UPDRS</td>
<td></td>
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<tr>
<td>Tremor score</td>
<td>1.0 ± 3.0</td>
<td>4.0 ± 3.0</td>
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<tr>
<td>Rigidity score</td>
<td>10.0 ± 2.5</td>
<td>6.5 ± 6.0</td>
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<tr>
<td>Bradykinesia score</td>
<td>8.0 ± 4.0</td>
<td>4.0 ± 3.5</td>
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MMSE = Mini-Mental Status Examination; BDI = Beck Depression Inventory; UPDRS = United Parkinson Disease Rating Scale.

Subjects sat at a desk in front of the digitizing tablet. They were required to move a pen-like stylus in a sagittal plane from a home position in front of the body midline, to a 12.5-mm circular target at a distance of 200 mm, giving the task an ‘index of difficulty’ of 5 according to Fitt’s law (Fitts and Peterson, 1964). The movement involved mainly arm anteversion and elbow extension. Subjects were instructed to make smooth single movements, which were both fast and accurate. Each trial started when the stylus was steady on the home position (a 5-mm dot). Movement started after two distinctive tones signalled ‘ready’ and ‘go’, with a randomly varying inter-signal interval between 1 and 3 s (instructions were different for the training with rhythmic guidance as described below).

The experimental sessions had a standardized structure: (i) 15 valid trials of baseline performance on the aiming task with each limb; (ii) 100 trials of training with one arm, with a short break after 50 trials; (iii) 15 valid trials with each limb; and (iv) a further valid 15 trials with each limb after a 1-h break. Clinical assessment was done during the 1-h break.

During training, subjects were asked to improve their speed of movement without losing accuracy. Knowledge of averaged results (Young and Schmidt, 1992) was provided after every five training movements as the mean percentage change in movement duration compared with the mean duration of the preceding five movements. Feedback for accuracy was provided only when the mean absolute spatial error across the five trials was >2 SDs above their individual mean spatial error during baseline.

While this basic structure applied to all subjects, half of the subjects received additional cues for the training trials. These consisted of five equally spaced tones. Subjects were asked to try to use the rhythm to control their movement speed, starting their movement after the third tone, reaching the target on or before the fourth tone and returning on or before the final tone. At the start of training, the frequency of the tones was set to 90% of the individual’s mean movement time at baseline. Thereafter, the frequency was adjusted after each block of five training movements to 90% of the mean of those five trials, thus setting a gradually improving target.

Data handling

Valid trials were those with an initiation time from the go signal of between 100 ms and 1000 ms, made in a single smooth movement to the target with no stops in between. Movements that did not fulfill these criteria were replaced for measurements at baseline, and after training. Movements were not replaced during training itself, where a fixed number of training trials was required. Invalid responses were not used for the calculations for ‘knowledge of averaged results’ or for adjusting the auditory cues.

Position data were smoothed with a seven-point moving median filter (Mottet, 1994). Differentiation was based on the seventh-order central difference method (Shakir, 1989). Various kinematic parameters were calculated. End-point accuracy was defined in terms of absolute spatial error in millimetres. In addition to total movement time, movement duration was measured separately for the early and late movement phases. These phases were identified by the point of maximum deceleration thought to define the end of the early (ballistic) phase and the start of the later visually guided corrective phase. Finally, maximum tangential acceleration and maximum deceleration were measured.

Statistical analysis of group data

As is usual, rapid performance change was observed over the baseline period. The first seven warm-up trials were discarded and the remaining eight trials used as an index of baseline performance prior to training. Mean scores were
calculated for each block of 10 training trials (T1–T10), for the test immediately after training (IT), and for the delayed test 1 h later (DT). Analysis of variance (ANOVA) for repeated measures was performed for each variable, using the MANOVA procedures of SPSS (SPSS Inc., USA). Averaged F-statistics were used with significance levels based on Huynh–Feldt adjusted degrees of freedom. The between-subjects factors were ‘diagnosis’ (patient or control group) and ‘condition’ (training with or without rhythmic cues). The within-subjects factor was ‘time’.

Results

Although the patients were bradykinetic (performance assessed by thumb flexions per 30 s), there was no evidence that they were becoming more impaired as the session proceeded [F(1,26) < 1]. Indeed, all sub-groups improved their performance between the baseline and IT assessments (Parkinson’s disease patients, uncued +4.9; Parkinson’s disease patients, cued +4.4; control subjects, uncued +5.6; control subjects, cued +5.6). There was no further change on testing at time DT.

The remainder of the results concerns performance on the aiming task. Because the task required subjects to improve speed without compromising accuracy, it was important to demonstrate that accuracy did not deteriorate across the session. No significant differences were found between the patient and control groups in the absolute spatial error at any point during the experiment. The mean absolute error of the groups remained within the range of 3–5 mm from the target centre throughout each experimental phase, and for both limbs. Similarly, the position at the end of the first movement phase did not differ between the groups. During training there was an overall tendency for this error to decrease in all groups (Parkinson’s disease patients, uncued –2.8 mm; Parkinson’s disease patients, cued –6.4 mm; control subjects, uncued –1.3 mm; control subjects, cued –2.8 mm). Accuracy will not be considered further.

Throughout the study, and as expected, the patients with Parkinson’s disease moved more slowly than the control subjects and had lower values of maximal acceleration and deceleration (see Figs 1 and 2). The one exception to the generally inferior performance of the Parkinson’s disease patients was in the duration of the final movement phase (see Fig. 1, unfilled upper bars). In contrast to the initial movement phase (Fig. 1, filled lower bars) which was prolonged in the patients [F(1,26) = 19.1, P < 0.001], the final movement phase was normal [F(1,26) < 1].

The main effects of diagnosis will not be reported in the subsequent analysis. Rather the results will focus on the task effects and any interactions with diagnosis. The main parameter of interest was movement time. Maximum acceleration and deceleration were highly coupled, and analyses produced qualitatively and quantitatively similar findings in almost all cases. Although both sets of data are presented in Fig. 2, only the results on acceleration will be reported.

Changes across training (T1–T10)

Changes across training were assessed by comparing blocks T1 and T10. Total movement time showed a significant overall decrease [F(1,26) = 51.9, P < 0.001] (see total bar height in Fig. 1). However, there was a differential effect of training condition, with a greater decrease in total movement time in the uncued condition (mean decrease across groups 182 ms) than in the cued condition (mean decrease 72 ms) [F(1,26) = 9.6, P < 0.01]. The interactions involving diagnosis were not significant.

The duration of the early movement phase decreased significantly across the blocks [F(1,26) = 17.9, P < 0.001], and to the same extent in the patients (~72 ms) and control subjects (~69 ms). However, there was a differential effect of training condition [F(1,26) = 5.5, P < 0.05] with a significant decrease being seen in the uncued (~110 ms) but not the cued condition (~32 ms). Both patients and control subjects showed this same pattern of results. The duration of the final movement phase also showed a significant reduction [F(1,26) = 22.8, P < 0.001], but to a similar degree across training conditions and diagnostic groups.

Maximal acceleration (Fig. 2) also increased across the training blocks [F(1,26) = 32.2, P < 0.001]. As with total movement time there was a differential effect of training condition [F(1,26) = 13.3, P < 0.01] with a greater overall increase in acceleration in the uncued condition. There was also a differential effect of diagnostic group [F(1,26) = 7.0, P < 0.05]. While both patients [F(1,13) = 14.8, P < 0.01] and control subjects [F(1,13) = 23.9, P < 0.001] showed an increase in maximal acceleration, only the control subjects showed a proportionally greater change in the uncued condition [F(1,13) = 9.8, P < 0.01].

Immediate and delayed effects of training: trained limb

Across groups, the total movement time decreased between baseline and IT [F(1,26) = 35.1, P < 0.001]. There were no significant interactions involving diagnosis or training condition. Across conditions, the patients showed a mean improvement in movement time of 150 ms and the control subjects 197 ms. The improvement for subjects who were trained with cues (177 ms) did not differ from that in subjects trained without cues (171 ms). A similar pattern of improvement was seen for the duration of the early movement phase [F(1,26) = 32.4, P < 0.001]. A differential effect of condition and diagnosis on change in the duration of the final movement phase was suggested [F(1,26) = 3.1, P = 0.09]. All groups showed a decrease in duration (control subjects, cued –67 ms; control subjects, uncued –83 ms; Parkinson’s disease patients, cued –72 ms) except for the
Training of aimed movements in Parkinson’s disease

Fig. 1 Movement time for Parkinson’s disease patients and control subjects, practising either with or without cues. Assessment phases are baseline (B), first 10 training trials (T1), last 10 of 100 training trials (T10), testing immediately after training (IT), and delayed testing 1 h later (DT). The filled bars show the early movement phase (mean and SEM), from the start of the movement until time of maximal deceleration, and the open bars show the late movement phase from that point to the end of movement.

Fig. 2 Maximum acceleration (open bars) and deceleration (filled bars) (mean and SEM) for Parkinson’s disease patients and control subjects practising either with or without cues. Assessment phases as for Fig. 1.
patients trained under the uncued condition who showed a small increase (+24 ms).

Maximal acceleration increased across groups between baseline and IT [\(F(1,26) = 42.8, P < 0.001\)], but more so in the control subjects than in the patients [\(F(1,26) = 8.2, P < 0.01\)]. Although the increased acceleration was somewhat greater in subjects not receiving the cued training, the effect was not significant. Figure 3 shows an example from an illustrative control subject and patient.

Retention of the gains over the delay period was assessed by comparing performance at DT and IT. A significant Condition \(\times\) Time interaction [\(F(1,26) = 4.4, P < 0.05\)] revealed that total movement time increased (43 ms) for groups who had received the uncued training condition, but it was essentially unchanged (–7 ms) for those who had received the cued training. None of the interactions involving diagnosis were significant, although the deterioration in motor speed was most evident in the control subjects. A similar effect was seen with changes in the duration of the early movement phase, although the interaction failed to reach statistical significance [\(F(1,26) = 3.5, P = 0.07\)]. There were no significant changes in the duration of the final movement phase over the retention period. Maximum acceleration showed a general decrease across groups [\(F(1,26) = 5.4, P < 0.05\)], with no specific effects of training condition or diagnosis.

Transfer to untrained limb

As with the trained limb, total movement time decreased between baseline and IT across groups [\(F(1,26) = 8.8, P < 0.01\)]. There were no significant interactions involving diagnosis or training condition, although the control subjects tended to show greater improvement than patients. Qualitatively and quantitatively, effects described for the trained limb were also present in the untrained limb at both IT and DT. Results of the analyses will not be given.

Discussion

Before discussing the effects of training, we will consider the kinematic characteristics of the aimed movement in the patients, as measured at baseline. Woodworth (1899) partitioned aimed movements into an early phase of ‘initial adjustment’ and final phase of ‘current control’. More specifically, an early movement phase (up to maximal deceleration) is considered preplanned or open-loop to some extent. In contrast, the final movement phase (after maximal deceleration) is thought to be more strongly influenced by sensorimotor integration during the movement (Carlton, 1981; Jeannerod, 1984; Beaubaton and Hay, 1986) and potentially corrective in nature (Carlton, 1980). In the present study this distinction is supported by a lack of correlation between duration of the early and final movement phases (\(r = 0.15\)).

With the Parkinson’s disease patients, the increased movement duration at baseline was largely due to a prolonged early movement phase, while the duration of the final phase was not statistically different from control subjects. Movement amplitude is determined by the magnitude of applied (muscular) forces and the timing of movement (Schmidt, 1988). In the present experiment, Parkinson’s disease patients did not have a problem with the spatial aspect of the early movement phase, as the distance travelled at the end of the early phase was normal. The basic motor problem of bradykinesia could therefore be one of either timing or force regulation. It is hard to distinguish deficits of these domains with movements of fixed amplitudes, where one domain is adjusted to changes in the other. It is therefore not surprising that the duration of the early phase was increased while maximal acceleration and deceleration (reflecting the effect of agonist and antagonist forces) was reduced. However, training effects indicated that deficits with Parkinson’s disease patients occurred predominantly in the force domain (maximum acceleration/deceleration), while training-induced changes in timing of the early phase seemed
similar to those of control subjects. This notion is in line with previous reports suggesting that, while Parkinson’s disease patients are relatively normal in their ability to vary the duration of their movements (Teasdale et al., 1990), they have problems in controlling muscle activation and force generation (Hallett and Khoshbin, 1980; Teasdale et al., 1990; Corcos et al., 1996). Correlational analysis did not favour the notion of a deficit in co-ordinating acceleratory and deceleratory activity, which could potentially affect the resulting forces; for both Parkinson’s disease patients and control subjects these parameters were highly correlated ($r = 0.92$ and 0.91, respectively). Consequently, a deficit in producing enough acceleration and deceleration can be assumed. Deficient force generation and scaling might therefore be considered as one primary deficit underlying bradykinesia.

**Motor learning in Parkinson’s disease**

In view of the importance of possible speed–accuracy differences, it is important to note that spatial errors remained largely unchanged, or even decreased, across the 10 blocks of training, in both patients and control subjects. Any improvement in movement duration, therefore, must reflect real behavioural gain and not just an adjustment in aiming strategy. The results clearly demonstrate that, with practice, patients with Parkinson’s disease can decrease their movement time in a visually guided aiming task. We will ignore the question of training method for the moment and consider the performance of the Parkinson’s disease groups in general. Across training conditions, the mean movement duration decreased from an average of 979 ms at baseline to 829 ms when assessed after training. This improvement of 150 ms compares with one of 197 ms in the control groups. The improvements in both groups occurred in both the early and late movement phases. The early phase was markedly slowed in the patients at baseline, with a clear reduction in the profile of maximal acceleration/deceleration generated. In contrast, the duration of the late phase was comparatively normal. Force generation, and the resultant movement duration of the early phase improved significantly with practice in the patients. Thus, it could be shown that even the defect of motor control thought to best characterize bradykinesia appears to be sensitive to the effects of practice and training. In comparison with control subjects, limitations could only be demonstrated for changes in the force-related parameters acceleration and deceleration.

When behaviour changes with practice, the distinction between performance effects and learning effects can be made (Schmidt, 1988). Performance effects may be due to non-specific factors such as familiarity with the task, changes in the level of attention or effort, or muscle warm-up. Learning as a more permanent change can only be inferred if improvement is maintained across time and/or transfers to different movement contexts.

Maintenance of the behavioural gains was assessed by comparing performance immediately after training with performance after a 1-h delay, during which the task was not performed. The results show that not all of the performance gains were retained. Movement duration increased and maximal acceleration/deceleration decreased. Because only 15 trials were given at time DT, it is difficult to determine whether the performance decline is an instance of the warm-up decrement typically seen in motor learning tasks after a break (Adams, 1961), or whether it represents a true failure to retain the full gains from the practice session. However, it should be noted that in neither patients nor control subjects had the values returned to baseline levels by the end of the 1-h delay. There was thus evidence of at least partial retention of the acquired skill.

Not only were there sustained improvements in the trained limb, but there was also evidence of improvement in the untrained limb in both patients and control subjects. Although of slightly decreased magnitude, they were qualitatively the same as those seen in the trained limb. This provides clear evidence of transfer of an aiming skill, at least between two homologous sets of effectors. However, more general transfer was not assessed. Thus, we cannot say whether or not there would have been any improvement if we had assessed the speed of wrist or finger extension, or the lower limbs on an analogous task.

Thus, there was evidence, at least by the criteria used in the present study, that patients with Parkinson’s disease are capable of improving their motor speed with practice, and that this improvement has the characteristics of a motor skill. While remaining slower than control subjects, the process of learning per se appeared to be relatively intact in the patients under these particular task conditions. More extensive training, and testing over longer delay periods, would be necessary to assess any eventual limits on the ability of the patients to learn and retain such skilled behaviour more fully.

How does the improvement of motor speed with practice fit with the other evidence of motor-learning deficits in Parkinson’s disease mentioned in the Introduction? It is perhaps notable that almost all of the research to date has focused on aspects of spatial accuracy or timing; improvements in movement speed have not been emphasized. Another difference is that the present task involved a simple single aimed arm movement, whereas most other studies have employed continuous motor tasks. Which one of these factors is the limiting factor in motor learning cannot be determined from current evidence.

**External auditory cues**

Half of the subjects were provided with auditory pacing tones at the target rate for that trial. It was predicted that such external cues would facilitate performance of the Parkinson’s disease group. The rationale was that Parkinson’s disease patients have problems in adjusting the speed of their movements if they are based solely on internal cues augmented only by average knowledge of
result. However, the effects of such cues were complex and counter to expectation. The greatest improvements in performance between the beginning and end of training were shown by the groups who practised without cues. This differential effect was seen in total movement duration, and duration of the early phase, but was most evident in the force-related parameters of acceleration and deceleration (see Fig. 3). While, in general, both patients and control subjects showed these differential effects of training condition, there was one exception. This was in the effect of force production. While the control subjects showed these differential effects of training condition, there was one exception. This was in the effect of force production. While the control subjects showed a greater increase of peak acceleration/deceleration across training in the uncued condition, this differential effect was reduced in the patients and statistically non-significant.

It is clear from these results that the provision of external cues was not an effective aid to training in the primary goal of this task (increasing the speed of aimed movements), nor did it serve to minimize the primary motor deficits in the patients. If anything, external cues interfered with the kinematic properties of the movement. One explanation for this effect is that they introduced an extra level of complexity to the task. Not only did the subjects have to be both fast and accurate, but they also had to try to synchronize their movements to an external signal. The extra demands of synchronization may have reduced the effort subjects were able to put into energizing and shaping the movement, resulting in smaller behavioural gains with practice.

One other possible effect of cueing was shown for retention over the delay. Although not always significant, the cued groups tended to show better retention of performance gains over the 1-h period. This might indicate that the aiming movements had become more skilled. However, longer follow-up periods would be necessary to determine the degree to which any acquired skill was preserved following the different training conditions.

Implications
Although primarily a laboratory-based investigation, the results may have implications for the clinical management of Parkinson’s disease. The primary result from this study was that patients with Parkinson’s disease were able, through practice, to reduce the level of bradykinesia in a particular task context. The level of performance reached during and after training was within the normal range defined by the 95% confidence interval of the control subjects’ performance prior to practice (Fig. 4). The improvements were, at least partially, transferred to the other, untrained limb, and the results were maintained during a period without practice. Such effects, for a fundamental deficit in Parkinson’s disease, provide the minimum basis on which to suggest that specific remedial strategies may be beneficial in reducing bradykinesia. Certainly, it is unlikely that training on a single task such as the one used here would have any major impact on real life skills unless they had very similar task demands. Nevertheless, the results of the present study illustrate that, with training, considerable improvements can be made with regard to bradykinesia. It seems possible that therapeutic benefit could be obtained with a specific programme targeting a variety of motor patterns involved in daily living.

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